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Micrococcus sedentarius bacteraemia presenting with haemophagocytic syndrome in previously healthy boy

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Abstract

Haemophagocytic syndromes are the clinical manifestation of an increased macrophagic activity with haemophagocytosis. Infection-associated HS was originally described by Risdall in 1979, in viral disease. Since the initial description HS has also been documented in patients with bacterial, parasitic or fungal infections. We describe a case of Micrococcus sedentarius bacteraemia in a previously healthy 10-y-old boy with haemophagocytic syndrome. Species of micrococci are generally considered as non-pathogenic commensals that colonize the skin, mucosae and oropharynx. We report the first case of Micrcoccoccus sedentarius bacteraemia in an immunocompetent host and first case of HS associated with Micrococcus species.

Introduction

The genus Micrococcus are generally considered as non-pathogenic commensals that colonize the skin, mucosa and oropharynx and are usually regarded as contaminants of skin and mucous membrane [1]. Nevertheless, cases of bacteraemia, endocarditis, ventriculitis, pneumonia, endophthalmitis, keratolysis and septic arthritis have been reported [2–9]. In these reports, micrococci were identified to be causative organisms, mostly in immunocompromized patients.

We describe a case of Micrococcus sedantarius bacteraemia with secondary haemophagocytic syndrome in a previously healthy 10-y-old boy. Our experience highlights first the need to consider all organisms as a potentially pathogenic if more than 1 culture yields them, and second that even commensal organisms could result in severe infection as in this case associated with haemophagocytic syndrome, which could be even fatal; thus there is a need to treat early and aggressively.

Case report

A 10-y-old boy was admitted to another hospital with history of high grade fever and non- confluent, non-pruritic maculopapular rash on the trunk and extremities, and abdominal pain that appeared 6 d before admission. He was referred to our hospital because of hepatosplenomegaly and bicytopenia. Physical examination revealed toxic appearance, fever (39°C), tachycardia, maculopapular skin eruption, servical submandibular, axillary and inguinal microlymphadenopathies, liver enlargement 1.5 cm and spleen enlargement 5 cm below the costal margins. On admission the patient's blood cell count disclosed bicytopenia and elevated alanine aminotransferase (ALT), aspartate aminotransferase (AST), lactate dehydrogenase (LDH) and C-reactive protein (Table I). During his follow-up, hepatomegaly was further increased to 7 cm below the costal margins and laboratory studies revealed that triglyceride, cholesterol and ferritin values were elevated (Table I).

Bone marrow aspiration which was performed to rule out secondary haemophagocytic syndrome, showed increased number of histiocytes with haemophagocytosis (Figure 1). Micrococcus sedentarius was isolated from 2 separate blood cultures on admission, and on the third d of hospitalization by BACTEC 9120 fluorescent system. From this system, secondary haemaphagocytosis due to Micrococcus species bacteraemia was diagnosed and ampicillin sulbactam was given. Immunoglobulins, lymphoid cell population on flow cytometry and blastic transformation studies were all normal. On the 7th antimicrobial therapy d, spleen was nonpalpable and both white blood cell and platelet count increased to normal levels (5000/μl and 364 000/μl

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Table I. Laboratory features.

WBC/PML (%)	3200/μl (40%)	EBV monospot test	Negative
Haemoglobin	10.3 g/dl	EBV EBNA IgM	Negative
Platelet	45 000/μl	EBV VCA IgM	Negative
ALT	138 U/l (N: 5–40)	EBV VCA IgG	Negative
AST	100 U/l (N: 8–33)	EBV EA	Negative
Triglycerides	1315 mg/dl (N <200)	HSV I IgM	Negative
Total cholesterol	250 mg/dl (N <200)	HSV I IgG	Negative
Fibrinogen	184 mg/dl (N: 188–430)	HSV II IgM	Negative
Ferritin	501 μg/l (N: 23–70)	HSV II IgG	Negative
CRP	7.02 mg/dl (N: 0–0.8)	CMV IgM	Negative
		CMV IgG	Negative
		Parvovirus PCR	Negative

respectively). ALT and AST level normalized at 14 d of antimicrobial therapy. During the follow-up, bone marrow aspiration was repeated and no haemophagocytosis was detected.

Discussion

Haemophagocytic syndrome (HS) is characterized by a systemic activation of macrophages/ histiocytes which are induced to undergo phagocytosis of haemotopoietic elements [10]. Haemophagocytic syndrome describes a clinical entity characterized by fever, severe constitutional symptoms, lymph node enlargement, hepatosplenomegaly, cytopenia and coagulopathy, liver dysfunction, and haemophagocytosis in the bone marrow, spleen or lymph nodes. Pancytopenia is the most common feature. There are 2 forms: a primary (hereditary) and secondary form that is associated with infections, as well as collagen vascular diseases and malignancies [10]. Infection-associated HS was originally described by Risdall in 1979, in viral disease [11]. Since the initial description, HS has also been documented in patients with bacterial, parasitic or fungal infections. Infection has been found to be associated with HS in half of all reported cases [12,13]. In patients developing pancytopenia, haemophagocytosis and disseminated intravascular coagulopathy in conditions of septic shock, HS triggered by bacteraemia should be considered. In fact, HS is associated with various types of disseminated bacterial infection, such as pyogenic bacteria (Gram-negative bacilli and Gram-positive cocci, Staphylococcus aureus, Streptococcus, Haemophilus, Serratia), Mycobacterium tuberculosis and other mycobacterial infections, Mycoplasma, Salmonella, Rickettsia, Ehrlichia, Chlamydiae (psittacosis), Brucella, Borrelia (Lyme disease) and Legionnella [10].

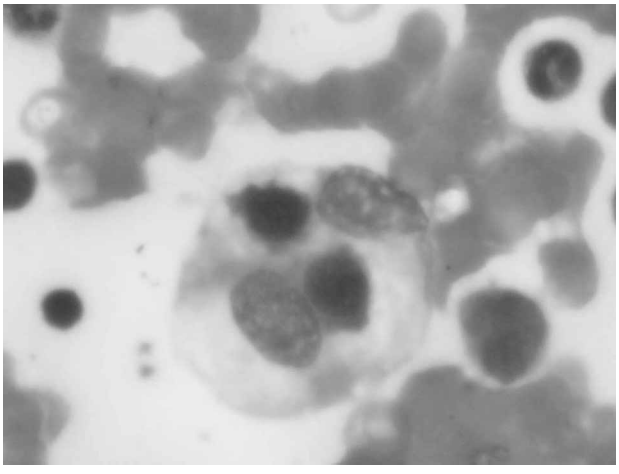

Figure 1. Wright stain of bone marrow aspiration shows increased number of histiocytes with haemophagocytosis.

Micrococcus species are usually regarded as non-pathogenic skin commensals. In immunocompromized patients, they could be opportunistic pathogens, and there are some reports of such cases [2–9]. In this patient, Micrococcus was initially seen as a possible contaminating microorganism. However, the absence of microorganisms other than Micrococcus species in several blood cultures, along with the repeated presence of microorganisms in the subsequent blood culture, suggests strongly that Micrococcus species were primarily responsible for the episodes of bacteraemia and HS.

We report the first case of Micrococcus sedentarius bacteraemia in an immunocompetent host and first case of HS associated with Micrococcus species. In summary, this report emphasizes that Micrococcus species should be considered as a pathogen even in an immunocompotent host in case of multiple blood culture results. It must be also emphasized that even non-pathogenic bacteria could play a role in the pathogenesis of HS; in such a case, the clinical course depends on the resolution of the underlying infection. Because HS is potentially severe and life-threatening, prompt recognition of the triggering agent is necessary to initiate appropriate therapy.

References

[1] Gahrn-Hansen B. Aetiological importance of coagulase-negative Micrococcaceae isolated from blood cultures. Acta Pathol Microbiol Immunol Scand B 1985;93:1–6.

[2] Adang RP, Schouten HC, van Tiel FH, Blijham GH. Pneumonia due to Micrococcus spp. in a patient with acute myeloid leukaemia. Leukemia 1992;6:224–6.
[3] Kim EL, Ching DL, Pien FD. Bacterial endocarditis at a small community hospital. Am J Med Sci 1990;299:87–93.
[4] Selladurai BM, Sivakumaran S, Aiyar S, Mohamad AR. Intracranial suppuration caused by Micrococcus luteus. Br J Neurosurg 1993;7:205–7.
[5] Magee JT, Burnett IA, Hindmarch JM, Spencer RC. Micrococcus and Stomatococcus spp. from human infections. J Hosp Infect 1990;16:67–73.
[6] Spencer RC. Infections in continuous ambulatory peritoneal dialysis. J Clin Microbiol 1988;27:1–9.
[7] Cartwright MJ, King MH, Weinberg RS, Guerry RK. Micrococcus endophthalmitis (Letter). Arch Ophthalmol 1990;108:1523–4.
[8] Nordstrom KM, McGinley KJ, Cappiello L, Zechman JM, Leyden JJ. Pitted keratolysis: the role of Micrococcus sedentarius. Arch Dermatol 1987;123:1320–5.
[9] Wharton M, Rice JR, McCallum R, Gallis HA. Septic arthritis due to Micrococcus luteus. J Rheumatol 1986;13:659–60.
[10] Tiab M, Mechinaud F, Harousseau JL. Haemophagocytic syndrome associated with infections. Baillieres Best Pract Res Clin Haematol 2000;13:163–78.
[11] Risdall RJ, McKenna RW, Nesbit ME, Krivit W, Balfour HH Jr, Simmons RL, Brunning RD. Virus-associated haemophagocytic syndrome: a benign histiocytic proliferation distinct from malignant histiocytosis. Cancer 1979;44:993–1002.
[12] Reiner AP, Spivak JL. Haematophagic histiocytosis: a report of 23 new patients and a review of the literature. Medicine 1988;67:369–88.
[13] Wong KF, Chan JKC. Reactive haemophagocytic syndrome: a clinico-pathological study of 40 patients in an oriental population. American Journal of Medicine 1992;93:177–80.

Mycobacterium marinum, a further infectious agent associated with sarcoidosis: The polyetiology hypothesis

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Abstract
A 39-y-old male had a diagnosis of sarcoidosis and corticosteroid therapy was started. Surprisingly, following his discharge from hospital, Mycobacterium marinum was isolated in 1 of 3 sputum samples taken 7 weeks earlier on admission. After this, Mycobacterium marinum-DNA was identified in the stored lung biopsies by the PCR-RFLP of the *hsp65* gene.

Introduction

Sarcoidosis is a multisystem granulomatous disorder of unknown cause(s). The diagnosis is established when clinicoradiological findings are supported by histological evidence of non-caseating epitheloid cell granulomas [1]. Granulomas of known origin must be excluded. Granulomas are organized collections of macrophages and other immune cells that arise in response to either a persistent intracellular pathogen, to a foreign body [2], or to an antigen of neoplastic origin [3]. A CD4/CD8 ratio >3.5 of the lymphocyte subpopulations obtained from bronchoalveolar lavage was found to provide a diagnosis of sarcoidosis with a specificity of 94% even if the transbronchial lung biopsy had not been diagnostic [1]. Spontaneous remissions occur in nearly two-thirds of patients, but the course is chronic and progressive in 10 to 30%. Most patients improve and stabilize under corticosteroid treatment but relapses occur in 16 to 74% of patients as the amount of drug is tapered or discontinued [1].

Many agents have been suggested as causes of sarcoidosis, but no firm consensus exists about the specific initiating agent: mycobacteria (Mycobacterium tuberculosis, M. avium complex, M. avium

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